

Myointimal Hyperplasia in Vein Collars for ePTFE Grafts

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Objective: To understand the cause of intermediate ePTFE graft failure when a venous boot is used. This might give insight into the improvement in patency that results from the use of a venous boot.

Design: A histological and radiological study of failed and failing grafts.

Methods: A review of patients operated upon in St Mary's Hospital under the care of a single consultant.

Results: Four anastomoses were retrieved following irretrievable occlusion and found to have myointimal hyperplasia at the junction between the ePTFE graft and collar. The artery was relatively spared. In a further six patients these results have been corroborated by angiography.

Conclusion: Myointimal hyperplasia occurs at the interface between ePTFE and vein collar and the recipient artery is relatively spared. This manipulation of the site of myointimal hyperplasia may result in improved patency rates.

Introduction

The superiority of autologous vein as the primary conduit in infrainguinal arterial reconstruction is now widely accepted. This superiority is most marked in infrapopliteal reconstructions, where prosthetic grafts have a particularly poor record. There is now some evidence (most recently from the Joint Vascular Research Group randomised controlled clinical trial¹) that a venous collar at the distal anastomosis significantly reduces the risk of failure in those patients whose lack of adequate lengths of vein condemns them to a prosthetic distal bypass.

Impressed by the patency rates reported by Miller *et al.* (21/29 grafts patent at a median follow-up of 18 months),² we adopted their anastomotic technique. Our encouraging early clinical results using this anastomosis have been previously reported.³

Our early success led us to conduct a number of *ex vivo* experiments to examine the putative haemodynamic advantages of this complex anastomosis.⁴ These experiments suggested various potential haemodynamic and mechanical advantages for the Miller collar and led to a modification of the technique⁵ but did not convincingly explain the clinical improvement

in intermediate patency. Others have developed further flow-rig experiments which are currently in progress.⁶

The reasons for the failure of these grafts have not been previously reported. We have therefore re-examined our ePTFE/collar graft failure in an attempt to understand the mechanism.

Patients and Methods

Only patients requiring femorotibial arterial reconstruction to ameliorate critical ischaemia,^{7,8} but without suitable autologous vein, are considered for prosthetic femorotibial bypass. In keeping with the philosophy of "vein if at all possible", bypasses involving the use of lengths of arm vein and venovenous anastomoses are used in preference to prosthetic reconstruction. Prosthetic bypasses comprise 21% of the infrapopliteal and 9% of all infrainguinal reconstructions and the unit has a 3% primary amputation rate.⁹ In common with the aggressive revascularisation philosophy advocated by Ouriel *et al.*,¹⁰ age and other medical problems are not in themselves considered a contraindication to attempted revascularisation.

The details of perioperative care have been previously described.^{3,5} The proximal anastomoses are

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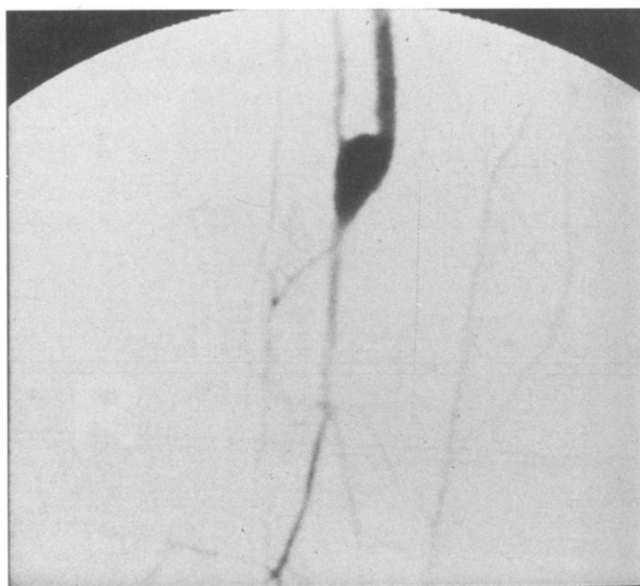


Fig. 1. Digital subtraction angiogram of patent ePTFE anastomosis to tibial artery using a venous boot.

performed in standard fashion with continuous side-to-end artery-to-ePTFE suture-lines. In patients operated on prior to April 1990, distal anastomoses were constructed as described by Miller *et al.*² After that date we used the modified St Mary's boot⁵ (Fig. 1).

All patients had 5000 IU of subcutaneous heparin twice daily whilst in hospital, followed by either aspirin 75 mg od or Warfarin (maintaining an INR at 2.0–2.5) indefinitely, depending upon age and compliance. All patients were entered into a programme of intensive graft surveillance. Thrombolysis was used for appropriate patients presenting with occluded grafts, with variable success (despite good radiological lysis our results of ePTFE are worse than either vein or Dacron¹¹), but the lysis gave us the opportunity to identify the cause of failure. Some irreversibly failed grafts have been retrieved for analysis following amputation. These have been dissected out, examined fresh, fixed and subjected to histological evaluation.

Results

We have had 15 immediate graft occlusions (i.e. failures of technique or judgement). Technical error at the anastomosis had been excluded by angiography, and it must be assumed that flow was lower than the critical thrombosis threshold for ePTFE.¹² Three patients had Buerger's disease – an association which led to permanent immediate graft occlusion in all cases. One of these anastomoses was retrieved but showed neither

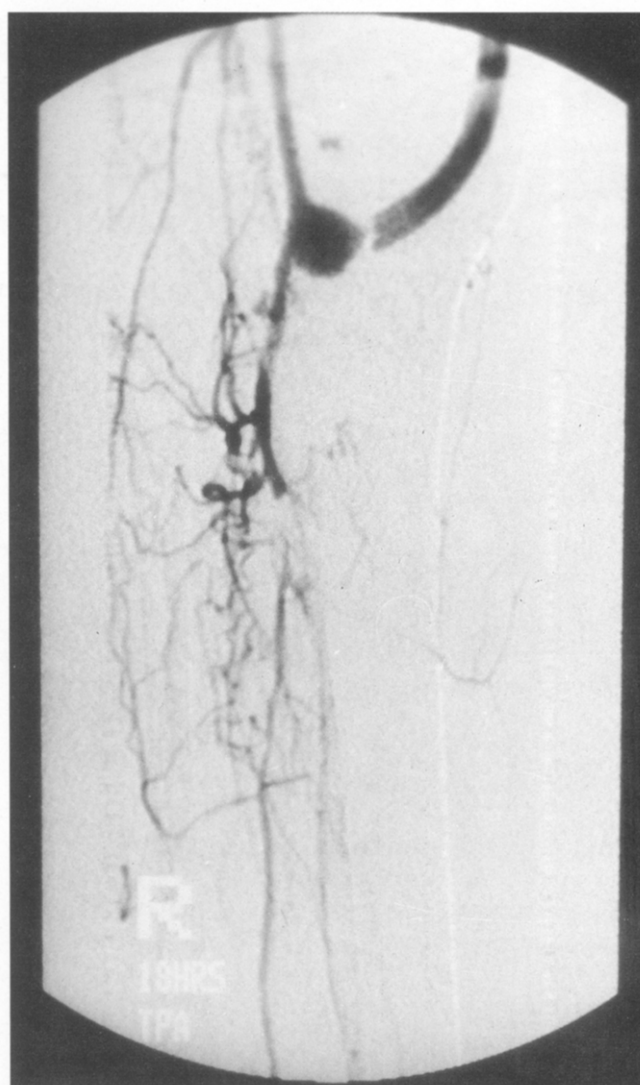


Fig. 2. Intra-arterial digital subtraction angiogram showing a tight stenosis at the ePTFE/venous collar anastomosis, following thrombolysis of an occluded graft.

technical error nor any special macro- or microscopic features.

Four anastomoses were retrieved following irreversible occlusion in the intermediate term. They represent occlusion at 1.2 months, 2.8 months, 5.8 months and 7.6 months. All demonstrated the macro- and microscopic features of intimal hyperplasia at the ePTFE/vein collar anastomosis with relative sparing of the recipient artery. In further patients, these findings have been corroborated radiologically. Following thrombolysis of an occluded graft, a tight stenosis at the ePTFE/vein collar anastomotic level was revealed as the cause of failure (Fig. 2). The hypothesis that the myointimal hyperplasia affects the ePTFE/autologous

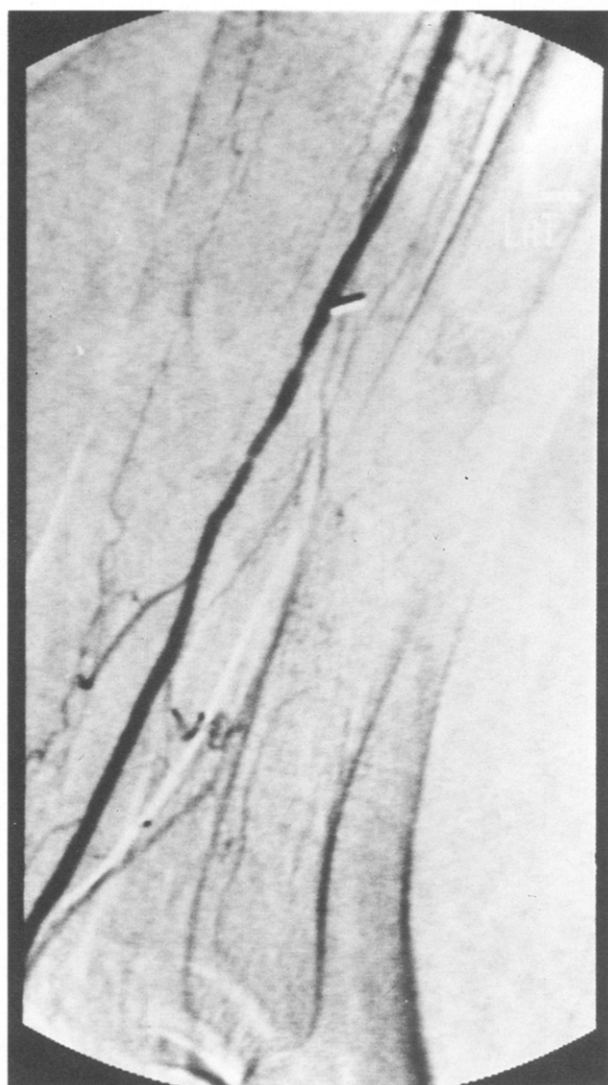


Fig. 3. Digital subtraction angiogram of a leg with an occluded ePTFE graft and venous collar, showing two indentations in the posterior tibial artery. These represent the anastomotic heel and toe.

anastomosis is supported by Fig. 3, where the site of anastomosis is marked by a clip. The recipient tibial artery has been largely spared by the process.

Discussion

At the time of its use at St Mary's Hospital, the Miller collar represented the evolution of a series of techniques aimed at facilitating the demanding anastomosis of relatively rigid 6 mm ePTFE to friable 2 mm tibial artery.^{2,13,14} Many surgeons around the world have been convinced by the advantages of the technique, and use it to ensure a technically sound distal anastomosis. One might expect this widespread utilisation to result in a reduction in immediate failure,

but there are those who believe that an adequate technical anastomosis can be achieved without resorting to the use of a vein collar.¹⁵ The fact that there appears to be an improvement in intermediate graft patency¹ needs a different explanation.

Anastomotic intimal hyperplasia might be expected to account for a high proportion of ePTFE graft failures.¹⁶⁻¹⁹ The histological and radiological evidence obtained suggests a benefit of the venous collar anastomosis. In particular, the myointimal hyperplasia is sited at the capacious ePTFE/collar anastomosis (an oval approximately 6 × 15 mm), where relatively large amounts of intimal hyperplasia are tolerated before causing graft thrombosis. This is important not only because the collar has greater tolerance for the accumulation of intimal hyperplasia than the small recipient artery, but also because sparing the recipient artery allows for secondary reconstruction in selected cases. The mechanism by which the vein collar redistributes intimal hyperplasia away from the critically narrow recipient artery is not known, but has been observed previously in an animal model.²⁰

Venous collars appear to improve prosthetic femoro-infrapopliteal patency rates, and this may be due to the ability to produce a technically sound anastomosis. Our analysis of failed grafts reveals another important factor, that anastomotic intimal hyperplasia accumulates at the capacious ePTFE/autologous tissue interface. The small recipient arteries seem to be spared.

We conclude that the venous boot improves prosthetic infra-popliteal graft patency rates by protecting the recipient artery from occlusion by intimal hyperplasia.

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